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ORIGINAL ARTICLES.

FILARIA LOA.*

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CINCINNATI.

ENTOZOA affecting the human eyes are rarely encountered in America, and of these rare guests the most unusual and in many ways the most interesting is the filaria loa. Henry B. Ward, Professor of Helminthology, University of Nebraska, writes me that in over one hundred references which he has carefully searched, he has found but one case where the parasite was actually extracted within the confines of the United States. My case is therefore the second on record in this country. It is claimed that there is only one small section of the world which produces the worm in question. My case was infected in that locality.

The patient, Mrs. A. B. Lippert, Westwood, Cincinnati, whose husband, Dr. A. B. Lippert, is a medical practitioner and missionary in western Africa, 135 miles from the west coast, in the equatorial region, in the country called Kamerun, first consulted me in August, 1901.

History: While residing in Africa in 1899, the patient one day noticed a prickling sensation in the right eye. The

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sensation was annoying, of the nature of an acupuncture, very positive, causing her to consult her husband at once in regard to it. Examination revealed the bulbar conjunctiva to be somewhat injected on the temporal side and the Doctor noticed a vermiform movement under the conjunctiva external to the sclera, looking very much like a worm crawling under the conjunctiva. At times the movement would cease for a few minutes, to be shortly resumed, "seeming very much like a snake forcing its way through a tangle of grass." In two hours time it had moved off the ball of the eye and escaped in the connective tissue at the outer canthus.

Her husband having heard that "eye worms" were common in that section of country, prepared some instruments for use in case the entozoon would manifest itself again. Two days later the same symptoms occurred in the left eye. The Doctor distinctly saw the worm crawling across the sclerotic of the left eye rather rapidly, and by the time he had the cocaine and instruments ready it had escaped. Within half an hour it appeared again in the upper eyelid between the skin and the tarsus. He made an incision with a knife over the point of greatest movement and succeeded in extracting the worm by means of dissecting forceps. Six months later another worm appeared in a similar position under the skin of the upper eyelid of the right eye. This time the knife severed the worm near its middle and one-half was extracted, while the other half remained in the tissues. The latter did not move afterward, but left a small nodule in the tissues which one can still see.

Three months after this a worm appeared in the neck over the sterno-cleido muscle on the right side, and could be distinctly seen. The operation was similar in technique, and the worm was extracted And again, eight months afterwards, a worm was felt and seen in the left mamma, about an inch above the nipple under the skin. This was attacked in the same manner, but was severed and one part removed, with no evidence of what ever became of the remaining part. On another occasion one was felt and seen at the angle of the scapula, but the instruments not being at hand, the worm made its escape.

On various occasions single worms were felt in the eyelid

and on the eyeball, her husband removing four at various times, but all were between the tarsus and the external skin of the eyelid. The patient remembers distinctly that the left ankle was affected with a worm, which produced considerable inflammation and œdema; and at the same time the right wrist was similarly affected, but the worms did not come to the surface, and both made their escape. The swelling gradually disappeared, leaving no bad results.

At various and numerous times the patient would feel worms at work near the joints of the body. The patient states that the insertion of a tendon was the principal point of irritation in every instance. Sometimes swelling, tenderness and œdema would accompany the irritation. Within a few hours the worm would be felt to change its position from one extremity of the body—for example, in the ankle—to some remote region like the wrist, of the other side of the body. At times the patient could trace the course of the worm from the ankle to the region of the ribs. The points of irritation would always be near the head of a bone lying near the skin where the worm passed between.

Examination: On August 9, 1901, the ophthalmic examination was negative; muscular apparatus in good balance; refraction +1.0 right and left; vision $^{20}/_{20}$, but patient has considerable asthenopia. There is slight catarrhal conjunctivitis and some blepharitis marginalis. No evidence of any worm was seen about the eyes, excepting the scars at various points, showing where her husband had operated for their extraction.

June 1, 1903. Patient returns, stating that she has again felt these eye worms on several occasions. At times they would be in the eyelids and at other times in the eyeball. Frequently they would be felt about the ankles, the ribs and the wrists. On one occasion she could feel a worm crawling across the sternum. It could be distinctly seen. Her husband being absent at the time, she called her brother, who attempted using a razor, but owing to his untrained surgical ability he only succeeded in making a slight cut, and both patient and surgeon losing confidence, the worm made its escape. She was endeavoring to have him sever it so that both ends would remain quiet and annoy her no further.

On September 17, 1903, while attending to her household duties she felt a worm creeping around on the right eye. Her husband brought her to my office, a distance of five miles, during which time the worm could be distinctly seen moving in the right upper eyelid. Examination at my office at first failed to reveal any evidence of the worm, but as the patient said it would probably manifest itself in a short while, I requested her to remain in my waiting room a few minutes in order to see if it would make itself visible. Before I could get ready to operate, in case the worm manifested itself, I was informed that it was again working near the surface. Examination revealed a distinct, independent movement, like what would be made by a small worm an inch long, crawling about under the skin of the upper eyelid. The movements were slow and somewhat clonic. During one of these contractions I grasped the most prominent loop of the worm by means of fixation forceps and then by means of scissors and forceps succeeded in extracting the worm without its being severed or wounded. It was placed in alcohol at once and carefully prepared for microscopical examination. The next day the Doctor called, stating that another worm had been seen on the other eye in about the same position, but the patient was not able to come to the office on account of the nervousness following the operation of the day before.

Four months later, viz: January 18, 1904, her husband called, stating that his wife could feel and he could see a worm creeping in the skin of the left lower eyelid that morning, but it was not convenient for her to come for its removal. Since last September the patient has felt these worms in various parts of the body.

It has now been two years and eight months since the patient left Africa, and these worms are as active as ever. She declares that she knows of four distinct worms still existing in her body which she feels at various times.

The Doctor is of the opinion that these worms at no time attack the vital organs nor the abdominal or pelvic viscera. They seem to limit their peregrinations to the extremities and especially the tissues about the eyes.

They can almost invariably be felt on chilly days out of season and cold days following a warm spell. If the patient

allows her foot or hand to remain from under the bed covering on a cold night the parasite can be felt.

Her general health is good; the vegetative functions are normal and there is no anæmia or cachexia. The patient does not attach much importance to this trouble, excepting that she is naturally anxious to get rid of it on account of her instincts and sensibilities being shocked.

Manson, in his book, "Diseases of Tropical Countries," has the following to say regarding this parasite:

"FILARIA LOA (page 517). This parasite is peculiar to the west coast of Africa, where, in many parts, it is not uncommon—in Old Calabar for example. The male worm measures about 25 to 30 mm. in length by 0.3 mm. in breadth; the female 30 to 40 mm. in length by 0.5 mm. in breadth. Both sexes are filiform, cylindrical, colorless, like fine fishing gut, tapering slightly at the head, more decidedly at the tail. The body is dotted over with minute chitinous bosses. The anus is subterminal. The tail of the male is markedly incurvated and provided with five large papillæ on each side of the anus, and two rather short unequal spicules. The mouth is simple, punctiform and without armature.

"The habitat of Filaria Loa appears to be the subcutaneous connective tissue, which it traverses freely. When, in the course of its wanderings, it crosses the subconjunctival connective tissue, and the delicate integuments of the orbit, it becomes distinctly visible. When passing under such thin structures as the skin of the eyelids, or that over the bridge of the nose, or when moving about under the skin of the fingers, the slight elevations it gives rise to, as well as its movements, are sometimes appreciable and may be both seen and felt.

"From the circumstance that this parasite has occasionally been seen in individuals who have been ten or more years away from the endemic area in which alone it can be contracted, we may conclude that the Loa is long-lived.

"The female produces embryos closely resembling filaria nocturna and filaria diurna. At one time I had a negro under my care, in whose blood filaria diurna abounded. This man remembered that as a boy he had Loa in his eye. On the other hand, Dr. Argyll Robertson has placed on rec-

ord a remarkable case of Filaria Loa, in which the parasites, a male and a pregnant female, were removed from the eye. The patient's blood was frequently examined but no embryos were found. Lately I had an opportunity of examining the blood of another patient known to be the subject of Loa infection, but failed to find any hæmatozoal embryos. We do not know anything about its life history outside of the human body. Notwithstanding the failures to find the embryo in the peripheral blood, analogy suggests that it probably circulates for a time in this fluid and, like filaria nocturna, that it is removed from this medium by some suctorial insect which acts as intermediate host.

"Filaria Loa, as stated, is very active in wandering about the body, but it seems to have a predilection for the eyes and their neighborhood. It comes and goes there at irregular intervals of days, weeks or months, traversing the subconjunctival tissue, and moving across the bridge of the nose from one eye to the other. Warmth seems to solicit its presence near the surface. The sexes seem to hunt each other about; at all events, in Dr. Argyll Robertson's case (*Trans. of the Ophthalmological Society*, 1895) a male worm was removed from the eye and not long afterwards a female worm appeared about the same situation and was also removed. The worm gives rise to pricking, itching, creeping sensations and occasionally, transient œdematous swelling in different parts of the body. When it appears under the conjunctiva, it may cause a considerable amount of irritation and congestion. There may be actual pain even, associated with swelling and inability to use the eye, and perhaps tumefaction of the eyelid.

"Treatment: The negroes treat Filaria Loa in two ways —either by dropping a grain of common salt into the conjunctival sac, which has the effect of driving the Filaria away, for a time at least; or they extract it with a sharp thorn, or by some other rude surgical means.

"With proper instruments there is no difficulty in removing the parasite from under the conjunctiva or from the eyelid—that is, if the operator is possessed of the requisite deftness in operating. All that is necessary, on the parasite showing itself, is, after cocainizing the eye, to seize the con-

junctiva with a pair of forceps, taking care to include the worm and the subconjunctival tissue in the grasp of the instrument. Having fixed the parasite in this way, the conjunctiva may be snipped through with scissors; when with the aid of another pair of forceps, and after releasing the first pair, the parasite may be withdrawn. In the case of the parasite showing itself elsewhere, I would suggest that it might be killed, as in the case of the guinea worm, by local hypodermic injection of bichloride of mercury solution (1 in 1000)."

In looking up the subject we find there are many varieties of nematodes, and there is some confusion in classification and nomenclature. Osler in his work on medicine recognizes three species of nematodes under the general term "Filaria Sanguinis Hominis."

(a) Filaria Bancrofti: the ordinary blood filaria (not the species found in my case). The embryos of this parasite are found in the peripheral circulation only during sleep (sometimes called Filaria Nocturna). The adult male worm measures over three inches, and the tail forms two turns of a spiral. The female measures over six inches. This is the species which causes Hæmatic Chyluria and Elephantiasis.

(b) Filaria Diurna: the larvæ resembling those of the Filaria Nocturna. The embryos are fouud in the peripheral circulation, only during waking hours. Manson thinks that the Filaria Loa represents the adult stage.

(c) Filaria Perstans: only the embryos are known. They are exceedingly small. Manson declares this worm is the cause of the "sleeping sickness" of negroes. There is a papillo-pustular skin disease found in West Africa called "craw-craw," said to be due to this parasite.

One variety has been observed in the anterior chamber of the eye of a horse (Turnbull, Phila., *Med. and Surg. Rec.*, 1878, p. 351). It grew to be four or five inches long, kept up a lively motion and at the last report was still growing. A few similar cases are reported where the worm, several inches long in each instance, was extracted and examined minutely.

Another variety of Filaria has been observed in the eye of man (loc. cit.).

Nordman of Odessa, as far back as 1830 and 1831, studied these worms, finding them in the eyes of cattle, sheep, pigs, frogs, lizards, fishes, birds and human beings. In fishes he first found entozoa in the vitreous, cornea, iris, retina and crystalline lens. This variety has been named "Filaria Lentis" and "Filaria Oculi Humani." Nordman examined two cataracts half an hour after they had been taken from an elderly man by Prof. von Graefe, in one of which he found two very small "thread worms." One of the two had been injured in the middle so that the intestines had come out of the body and were visible as slender threads. The other was uninjured, of uniform thickness, ¾ line long ($^1/_{16}$ inch), extremely narrow. It was spirally convoluted and dead. A simple intestinal canal, a mouth with visible papilla, a uterus and a prominent anal aperture could be distinguished." (Nordman, 1831, Mic. Beiträge, z. Nat. d. Wirb. Thiere, pp. 7, 8). In 1832 he investigated this subject still further and found the same worms in cataracts in several instances (loc. cit.). Von Ammon has corroborated these discoveries in a case of cataract which he extracted (*Klin. Darstellung*, Vol. XII).

In a chapter on Entozoa (Norris & Oliver) by Salzman, we find a description of the variety of worms illustrated in my case of filaria loa. I quote the following: "We know but little of the natural history of the Filaria Loa. The Filaria Diurna, that by day is found in the same places in great numbers in the blood of negroes is supposed to be the embryo of the Filaria Loa. It is believed that these embryos are sucked up with the blood of the negroes by the mosquitoes, that they undergo further development in these intermediate hosts, and that they are deposited in the water with their eggs. Water contaminated in this way may cause reinfection of man." He further states, "Its acclimatization is not to be feared, since the conditions favoring its propagation are present only in a circumscribed area of its normal habitat (West Africa)."

In the March number of the *Ophthalmic Record* we find an illustrated review of an article by Ransom on "Manson's Eye Worms of Chickens." The illustrations correspond with those of the true Filaria Loa, but the worms do not live

in the connective tissue as illustrated in my case report. They exist under and around the nictitating membrane in the bird, where they lie coiled, and can be extracted in great numbers. They infect the nasal passages of the fowl later on and cause death. They also cause the eye to suppurate. They are found in this country, especially in Key West, Florida.

I have the privilege of presenting the complete bibliography on the subject, compiled by Prof. Henry B. Ward, Lincoln, Nebraska, who has recently written a work on the subject which will attract widespread attention at home and abroad by all interested in Helminthology.

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SOME EXPERIENCES WITH ADRENALINE.

By H. M. POST, M.D.

ST. LOUIS, MO.

THERE is something very pleasing about the immediate effect of adrenaline upon the eye. It is pleasing to the physician to get such a prompt and visible result from an application, and it is very pleasing to the patient to see an inflamed eye, which he has watched by means of a hand mirror carried in his pocket, suddenly become pale and clear like its unaffected fellow. It also gives him confidence in the mysterious power of his physician over disease. On both sides, physician and patient, the effect is fine.

I think some such motives have influenced many of us in the use of this drug. There are, however, good reasons for its use, and there are certain bad effects from its use. I experienced the latter from the use of its equivalent, suprarenal extract, a little over eight years ago, in the early days of this class of drugs, which made me feel that they were very dangerous and resulted in my abstaining from their use till a short time ago, and I am very careful in the use of its successor, adrenaline, at present.

Adrenaline has a very positive effect in prolonging cocaine anæsthesia and in controlling hæmorrhage in operations about the eye; also in controlling hæmorrhage, and producing anæsthesia used with cocaine hypodermatically, in operations involving removal of tumors, and in plastic operations.

I have also found that in eyes where, on account of conjunctival congestion, cocaine was not absorbed, or atropia did not dilate the pupil, the use of adrenaline, by diverting the blood from the conjunctival vessels and producing an ischæmic condition of the tissues, would result in the absorption of these drugs and in their producing their normal effects.

In the treatment of hay fever conjunctivitis, I have found adrenaline, added to the usual treatment in such cases, of great benefit, the patients telling me that the attacks were prevented, cured or made much less severe than usual, and they consider me quite a hay fever doctor. I think it is possible that through the tear ducts portions of the affected mucous membranes are reached, which the treatment of the

rhinologist does not touch. In this connection I would add, that in anæsthetizing the nasal duct for the passage of probes, I have to avoid the use of adrenaline in some persons, as its use with them is always followed by a severe headache, which from their description seems to be similar to that caused by closure of the passage from the frontal sinuses into the nose.

My first unpleasant experience following its use—and with my later knowledge I have thought possibly depending on it—was in a case of a young lady where I did a tenotomy with an advancement. The pain during the night following the operation was so severe that it was necessary to give her morphine freely. I have done quite a number of similar operations and this was the only case in which morphine was given.

My first serious result, and the one which opened my eyes to the fact that suprarenaline was not the innocent drug we had thought it to be, but that its use was open to dangers we had not suspected, occurred November 9th, 1898.

A lady from out of the city, 38 years of age, who had been a patient of mine previously, called upon me with a large chalazion, lower lid, near outer canthus, O.D. I used cocaine 5 per cent. and infusion of suprarenal capsule freely before and during the operation. The operation was done in the usual manner, opening and scraping out the sac, and the patient left my office in good condition. This occurred some time in the forenoon.

In the early part of the afternoon, probably two or three hours after using the cocaine and suprarenal capsule infusion, she returned, with intense pain in the right side of her head, veins and arteries in the right temporal region very much dilated, and, as I recollect (I have no note of it), the pupil widely dilated. This latter condition would have been expected as a result of the use of the cocaine.

I will admit that I was very uneasy, and I gave her my undivided attention. I used hot water applications constantly, and at the end of about two hours of unsuccessful effort, I administered five grains of phenacetine, which gave her relief. It was two and a half hours from the time I began to use the hot applications till the patient was relieved.

This experience was a very severe lesson to me, and I dropped the use of the infusion of suprarenal capsule and did not use any of its equivalents for four or five years, notwithstanding the beautiful results which were published by numerous gentlemen, obtained by its use in glaucoma, iritis, etc. I thought I had not talent enough to handle it properly, and that I would depend on remedies that I knew more about.

My second instructive case, which again warned me in regard to its use, occurred recently. Another lady, aged 44 years, whom I have had under my care for glaucoma of the right eye since December 12th, 1904, but, from the history which she gave, the disease had probably existed for several years previously, was doing very well except for an annoying and profuse overflow of tears, with considerable hypertrophy of the conjunctival papillae. This hypertrophy was present on the lids of the left eye also, but to a very slight degree. I believe its excessive amount on the lids of the right eye was due to the irritation from eserine and pilocarpine, which was used in the treatment of that eye. The pupil remained smaller than that of the left eye; tension and vision normal; very slight limitation of the field.

In two cases of excessive lacrimation, from exposure to cold and wind, I had had excellent results from the use of adrenaline $^1/_{1000}$, followed by zinc sulphate gr. j to ʒ j. From this experience I thought this might be a good line to follow with my glaucoma case. I used it in my office carefully, with no unpleasant effects, for four days. I then (Oct. 2nd, 1905) prescribed adrenaline $^1/_{2000}$, to be followed by zinc sulphate gr. j. to ʒ j. This was used daily by the patient till October 6th, when during my absence from the city the patient came to my office and was seen by my assistant, Dr. Shahan, and by Dr. Ewing.

Dr. Shahan reported as follows: "Patient used gr. $^1/_{240}$ O.D. this morning and about an hour afterward used adrenaline $^1/_{2000}$ prescribed for her October 2nd, 1905. This was followed 35 minutes later by a dizzy sort of faintness and wide dilatation of the pupils. Used Es. gr. $^1/_{960}$, with no effect at the end of half an hour. When seen at office, pupil O.D. was still larger than that of O.S., but gradually contracted to size of pupil of O.S., about one and a half hours after

Es. $^1/_{960}$ had been used. O.D. T+. O.S. T. With glasses O.S., everything looked dim. V=$^{20}/_{24}$."

The day following I saw the patient, at which time the "pupil O.D. was the same size as that of O.S. V=$^{20}/_{12}$ either eye. Es. $^1/_{240}$ (very minute particle, about $^1/_{10}$ of a minim.) produces strong miosis, and reduces the Hm. from 2.5 D. to 1.5 D."

Adrenaline has not since been used, and the case is progressing favorably.

Now I come to my third and last experience, by far the most interesting, of positive value, and I do not know of any similar observations. The patient is a lady, 64 years old. For the present trouble I first saw her the 12th of last May. The record at that time reads as follows: "Two months ago had severe pain in O.D. The first day of this pain O.D. closed." She was confined to her bed for a time, and my report goes on to say, "Yesterday was the first time she has been able to be up all day. Present condition, ptosis, and paralysis of the third nerve O.D. O.D. Hm. 3.5 D.V=$^{20}/_{15}$ to $^{20}/_{12}$. O.S. Hm. 2 D.V=$^{20}/_{15}$ to $^{20}/_{12}$

"May 18th: Pupil O.D., about normal in appearance, but does not respond to either light or accommodation. O.D. with + 3. V=$^{20}/_{30}$ +. Homatropine gr. $^1/_{240}$ dilates pupil O.D. fully, after which ophthalmoscope gives eye ground normal O.D. and O.S."

June 19th, she disappeared. She was under the care of Dr. Frank R. Fry, and her visits to me were only for observation, and were more or less irregular. She seems to have gone home, as she returned to me September 20th, her physician having written Dr. Fry that in his opinion O.D. should be removed, and Dr. Fry had sent her to me with the statement that her trouble was nuclear in origin. My notes read as follows:

"Has had no severe pain, but almost constant dull pain in O.D. since here. O.D. with + 3. D.V=$^{20}/_{192}$. O.S. Hm-3. D.V=$^{20}/_{30}$ to $^{20}/_{24}$. Dilated pupil O.D. with homatr. gr. $^1/_{240}$; cornea hazy; some keratitis lower portion.

"September 22nd: Lower portion of cornea anaesthetic and ulcerated."

The local treatment from that time has been cleansing

the eye, the use of antiseptics and keeping the eye closed with a strip of gauze fastened with contractile collodion.

I have gone thus into detail in order that you may be in a position to estimate fully the value of the following observations.

In treating the ulcerated cornea I used adrenaline, followed by zinc sulphate, not noticing the size of the pupil before the adrenaline was used. I noticed, however, after using it that the pupil was larger than the corresponding one. Upon watching the pupil more carefully, I assured myself that the pupil dilated after the use of the adrenaline solution, and this led to my making the following measurements:

December 5th, 1905, I dropped into the eye a solution of adrenaline $^1/_{2000}$, the same solution that had been used in the case of glaucoma. Before using the medicine the pupil was 3 mm.; one hour later it was 6 mm.

December 7th, thinking that there might be something peculiar about that particular solution, I used a drop from an original bottle $^1/_{1000}$. Before using the medicine the pupil was 3 mm.; an hour and a quarter afterwards the pupil was 5 mm.

December 9th, I again used adrenaline $^1/_{1000}$ from the same original bottle, making frequent observations in order to get some idea when the maximum effect was produced. When the drop was used, the diameter of the pupil was 3 mm.; 21 minutes afterwards, 5 mm.; 30 min., 5 mm.; 46 min., 5 mm.; 1 hour, 4.5 mm.; 2 hours, 3.5 mm.

I would add that at my request, my assistant used adrenaline $^1/_{1000}$ in at least three cases with normal eyes, producing no mydriasis.

The drug used was solution adrenaline chloride, Park Davis & Co.

RETINAL CHANGES AS AN AID TO DIAGNOSIS IN VASCULAR DEGENERATION.*

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WE have in the eye the only organ where a terminal set of vessels can be seen and where it is possible to observe by means of the ophthalmoscope changes in the retinal vessels and capillaries that enable us to detect in many cases alterations that are indicative of disease of the general vascular system. The information thus elicited is important from a clinical standpoint, in that in many instances it is the first inkling of the fact that degenerative changes are taking place elsewhere, and those seen in the eye are only part of a morbid process existing in the blood and bloodvessels of the brain, kidneys and other portions of the body.

It is not my intention in this paper to speak so much of gross lesions occurring in the background of the eye, whether of the retina, choroid or the optic nerve, as to call attention to those slight anomalies that are recognized from time to time and show themselves in alterations in the caliber and contour of the retinal vessels, sometimes so slight that they may be easily overlooked if a careful and diligent study of the background of the eye is not given.

We all know that certain abnormal conditions of the fundus depend for their existence upon pathological changes, usually of a degenerative character, going on in other organs of the body, having for their origin a diseased condition of the blood and bloodvessels. These occur in people who are apparently in good health; nothing of a suspicious nature for one moment leads us to think that anything is wrong until an ophthalmoscopic examination is made when the patient consults the ophthalmologist for some apparently trifling symptom referable to the eye.

We are enabled by closely studying the fundus to detect very slight structural changes in the retinal vessels which

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lead us to form conclusions of an almost positive nature that there is a commencing degeneration of the arterial walls which are suggestive of the existence of a similar condition of affairs in the smaller arteries situated elsewhere in the body and signify an arterio-capillary fibrosis.

Tortuosity of the retinal vessels, although one of the earliest phenomena, is not in itself a positive sign unless confirmed by the presence of other variations from the normal which are generally to be found in the early stages of arterial degeneration. These are the alterations in the caliber of the vessels showing inequalities in the breadth of the vessel at various points in its course. The localized contractions in some instances amount to almost total disappearance of the capillary which then regains its normal size; this condition occurring over and over again, giving the vessels a bead-like appearance. At the same time the vascular reflex is broader and brighter and whitish stripes run along the margin of the vessel wall.

Loss of translucency, in which the arteries lose their power of transmitting light, occurs where a vein is crossed by an artery. The vein becomes obscured at this point and the phenomenon is usually accompanied by an appearance of compression and loss of the light streak in the vein on either side of the point of crossing. In the more advanced stages the vein is practically obliterated from the pressure exerted by the artery, and enlargement of the distal end of the venous current is quite apparent. On the other hand, when the vein crosses the artery it appears to hook over that vessel with less signs of compression of its coats.

Oedema of the retina from interference with its nutrition occurs sooner or later and is recognized as a grayish haze, which is better seen in the macular region or along the course of the vessels. Hæmorrhages into the retina eventually show themselves as the disease advances. These changes occur along the course of the vessels or at isolated spots in the retina, or they may even be sub-hyaloid in character.

Microscopically these changes are found to be due to a thickening and loss of elasticity of the walls of the vessels and to a narrowing of their lumen from the formation of connective tissue, mainly in the intima. These degenerative

alterations effect both the muscular and elastic tissues and eventually bring about hyaline degeneration of the entire vessel wall. The pathologic changes are never really primary in character, but are a part of a general arterio-capillary fibrosis, which has been gradually developing for a variable length of time as the result of the absorption of an endogenous poison produced by some derangement of nutrition.

The eliminating powers of the excretory organs become interfered with and the toxic elements are absorbed into the blood, producing an irritation of an infective nature. The relation of vascular disease to various morbid conditions in certain organs of the body is well known. The causes that lead up to these changes in the circulation depend upon the absorption into the blood of the toxins given off under diseased conditions existing in the general system, which, if not checked, have an effect upon the vascular coats, that ultimately result in pathological changes which sooner or later become permanent and produce degenerations in various organs of the body. They are due to the retention in the system of effete substances which act as poisons, their existence depending upon defective action of the excretory organs.

If such excretory organs as the kidneys and liver, act indifferently there is an accumulation of toxic matter which absorbed into the blood stream produces morbid changes, not only in the vessels of these organs, but more particularly in the general vascular system.

Primary arterio-sclerosis depends in great measure on just such a cause. Arterio-sclerosis in the true sense of the term is probably never of primary origin, but is secondary to a degeneration which has already existed for some length of time. The patients whose retinal vessels show these degenerative changes are generally those who have reached the age of forty or fifty years, who have led a sedentary life, and who are fond of good living. There is, also, a history in most instances of some form of "indigestion," and in many cases there is a distinct history of gout or "rheumatism." It is in such cases that the natural functions of the excretory organs are not carried on perfectly. A form of poisoning results from absorption into the blood of the effete matters which are insufficiently eliminated.

Gout, syphilis and alcoholism are associated very frequently with, and play an important role in the causation of these vascular degenerations. They are very likely in the majority of instances the underlying cause of the arterial disease. What effect the poisons have upon the blood and the vessel wall is not clearly understood, but these changes are probably of such a character as to cause a high blood pressure from increased resistance to the blood current in the peripheral vessels. This is followed by hypertrophy of the heart and eventually atheromatous changes in the arteries. As the disease advances in the system and a general arteriosclerosis develops, the changes in the retinal vessels may be regarded as merely a part of those vascular alterations that are at the same time taking place elsewhere in the body. These pathologic changes may go on for years without giving rise to any untoward symptoms that have an effect upon the patient's ordinary mode of life. It is only when the disease has advanced to the stage in which foci of sclerosis appear in the kidneys, liver and the larger vessels that notice is taken of them or even a suspicion is entertained that the patient's health is effected and that his life is in danger.

There is probably no other disease in which arteriosclerosis plays a more conspicuous part than atypical chronic gout, a disease characterized among other changes by a high arterial tension and cardiac hypertrophy. The gouty diathesis predisposes to vascular degeneration of a more or less pronounced type, the presence of which is recognized in retinal changes confined more or less (especially in the early stages) to certain alterations in the retinal vessels and circulation, although these changes do not differ materially from those which are present in the course of arterio-sclerosis due to other causes. It is well recognized that degenerations of the retinal vessels, of the retina itself, are probably more indicative of chronic gout than any other morbid process. This is probably due to the presence of deleterious matter in the blood, which, acting on the coats of the vessels induces a high arterial tension, and is responsible for the alterations in the arterial walls themselves. These same causes bring about sclerotic metamorphoses in the smaller bloodvessels and capillaries.

The degenerative changes show themselves in the retina by alterations in the appearances of the bloodvessels which in the early stages are so slight that they are easily overlooked if a careful examination is not made. The retinal arteries are diminished in caliber with more or less tortuosity of the distal end of the vessel; the bright streak is broader and there is decided loss of translucency, so that the underlying vein at a point of crossing does not show itself through the artery but seems pressed upon almost to the point of obliteration.

As this degeneration becomes more extensive œdema of the retina, even to the extent of interference with the visual power, develops. Later on, in the more advanced stages of the sclerosis, the vessels become engorged and tortuous, and hæmorrhages take place from the degenerated capillaries.

PROGNOSIS.

The significance of these retinal changes and the influence they exert upon the life of the individual depend largely upon their cause and the progression of the degeneration in other portions of the body. The degenerative process which so often has an insiduous beginning may have gone on for some time before any abnormalities in the ocular fundus are noticed. Not until gross lesions have shown themselves in the eye can we positively say that a fatal termination is apt to occur. The mere fact that such conditions exist in the retinal vessels, does not warrant us in assuming that the disease is going to progress to such a stage as to effect the function of the kidneys and liver or to cause degenerative changes in the brain.

The course of arterial degenerations is usually a slow one, although exhibiting periods of exascerbation. The patient's ordinary occupation is very little interfered with. There are ordinarily no symptoms of any moment which would point to an altered state of the general health. The patient lives his daily life unconscious of the existence of these phenomena until some marked symptoms from the occurrence of retinal hæmorrhages, thrombosis of the retinal vessels, etc., show themselves and the clinical aspect of the case becomes decidedly changed. When these vascular changes are recognized and appropriate treatment is instituted early, the progress of

the disease may be arrested and the patient's health remain apparently in a normal condition. An unfavorable prognosis depends upon the state of the heart and other organs. When the degeneration has so far advanced that the vascular walls are impregnated with tissues that are foreign to them and hæmorrhages into the retina and obstructions in the circulation have occurred, then an arrest of the progress of the disease cannot be hoped for. The main points I desire to bring out in this paper are embodied in the following conclusions:

1. The retinal vessels are liable to changes which are indicative of general vascular disease.

2. That these changes are readily recognized by ophthalmoscopic examination in the very early stages of the arterial degeneration.

3. When recognized early and appropriate treatment is applied, the disease can be arrested and a favorable prognosis given.

4. The principal early ophthalmoscopic signs are, (a) contraction of the arteries; (b) compression of veins where crossed by arteries; (c) loss of translucency of the arteries; (d) broadening of the reflex streak along the course of the vessels.

EDITORIAL NOTICE.

We are asked to state that an investigation as to the HEREDITY OF ALBINISM IN MAN is being carried out by Professor Karl Pearson, F.R.S., of University College, London (Eng.), with the co-operation of Mr. E. Nettleship (Shottermill, Surrey, Eng.), and Dr. E. Stainer (Physician to the Skin Department, St. Thomas' Hospital), 60 Wimpole St., W., London (Eng.), and that cases bearing on the subject, *either positively or negatively*, will be very thankfully received by any of those gentlemen. The investigators desire to collect a large series of examples of Albinism—complete or incomplete, general or partial—in which the family history of the albino patient can be traced for one, two, three or more generations. The sex and prevalent color of eyes and hair in as many of the patients' relations and ancestors as possible should also be noted, whether any of them were albinotic or not; also all instances of consanguineous marriage. Cases of albinism of the eyes in persons with congenitally pied skin (most easily recognized amongst dark races) will also be very valuable even without family history—acquired leukoderma being of course excluded.

INDEX TO VOLUME XXII.

ORIGINAL AND SELECTED ARTICLES.

PAGE

Adrenaline, some experiences with 370
Advancement of recti muscles 83
Amblyopia, unique cases of 51
Argyrol, experience with staining of the skin 86
Aseptic and antiseptic work prior to operations 10
Bacillus pneumoniæ in conjunctivitis 1
Bacillus pyocyaneus in conjunctivitis 4
Blank cartridge wound of eye 5
Cataract, new attempts at cure without operation 202
Choroiditis, central superficial 29
Ciliary processes in accommodation 324
Clamps for fixation of eyelid 88
Coffee amblyopia 55
Complications following cataract extraction 8
Complications and accidents met with in cataract extraction .. 330
Dionin 39
Ectopia lentis with apparent accommodation 131
Expert testimony in ophthalmology 259
Exudate into the anterior chamber, unusual, after extraction .. 236
Eye cases, report of several 142
Filaria loa 355
Foreign bodies, metallic, in the eye and their removal 97
Glaucoma simplex with myasthenia gastrica and intestinalis .. 318
Gumma of the optic nerve 199
Gumma of the optic nerve and choroiditis gummosa 228
Intradurnal tumor of optic nerve 177
Iridocyclitis albuminurica 170
Iris, double rupture of 138
Iritis, two cases in Bright's disease 195
Keratoconus, treatment of 73
Lens, double coloboma of 147
Lens capsule in the operation of cataract extraction 262
Mammalian eye, fundus appearances 291
Mules' operation, technique 82

Nitric acid, pure, in treatment of the eye 68
Papillo-epithelioma, epibulbar 242
Pinguecula and pterygium 268
Retrobulbar neuritis, two cases of 163
Retinal changes as an aid to diagnosis 375
Semaphore charts 33
Strabismus, Panas' operation 208
Test type construction, principles of 79
Thiersch grafts 83

SOCIETIES.

American Academy of Ophthalmology and Oto-Laryngology, 252, 289
American Ophthalmological Society 161
British Medical Association 284
Ophthalmological Society of the United Kingdom, 92, 149, 175, 249

ABSTRACTS.

Accommodation, subnormal and premature presbyopia 254
Annular keratitis 186
Asthenopia and eyestrain independent of refraction 188
Blindness and oculomotor palsies from injuries 226
Cancer of the choroid, metastatic 179
Contagious, acute, inflammations of the conjunctiva in children 181
Diaphoriesis and diaphoretic agents 180
Entropium, senile, simple operation 155
Errors, slight, of refraction 290
Esophthalmus, differential diagnosis 182
Eye affections in sinus 185
Eyestrain, its importance and limitations 256
Eye symptoms in general disease 184
Haab's giant magnet 156
Hæmorrhage, intraocular 155
Heredity and treatment of squint 157
Loss of visual power in school children 159
Nervous symptoms in children 179
Obstruction of retinal arteries 289
Ocular therapeutics, advances in 189
Ophthalmology, the new 154
Strabismus a clinical and statistical study 159
Strabismus, operative treatment 187

Test of muscle balance for near and distant vision..... 290
Thromposis, septic, of cavernous sinus 225
Trachoma, non-operative treatment 160
Tubercle of the choroid 150
Visual acuity, comparative, of savage and civilized people.... 156
Visual field, contraction of..... 226
X-Ray treatment of external eye diseases 186

REVIEWS.

Book reviews 174, 191, 298

PAMPHLETS.

Pamphlets 197

CORRESPONDENCE.

S. M. Burnett 96

EDITORIAL NOTICE.

Editorial notice.....174, 267, 380

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CONTENTS.

ORIGINAL ARTICLES. PAGE

Filaria Loa. By DERRICK T. VAIL, M.D., Cincinnati 365
Some Experiences with Adrenaline. By H. M. POST, M.D., St. Louis, Mo 370
Retinal Changes as an Aid to Diagnosis in Vascular Degeneration. By THOMAS A. WOODRUFF, M.D., Chicago, Ill 375

Editorial Notice 380

Index to Volume XXII 383

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